1. A method for identifying a candidate compound that modulates lipopolysaccharide (LPS)-mediated activation of NF-kB, the method comprising:

providing a cell that harbors LPS and expresses a polypeptide comprising a caspase recruitment domain (CARD), nucleotide binding site (NBS), or leucine rich repeat (LRR) domain of CARD-4;

exposing the cell to a test compound; and measuring NF-kB activation in the cell;

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wherein altered NF-kB activation in the presence of the test compound compared to NF-kB activation in the absence of the test compound indicates that the test compound is a candidate compound that modulates LPS-mediated activation of NF-kB.

- 2. The method of claim 1, wherein the cell is infected with Shigella flexneri.
- 3. The method of claim 1, wherein the cell is infected with Salmonella typhimurium.
- 4. The method of claim 1, wherein the cell is infected with Helicobacter pylori.
- 5. A method for identifying a candidate compound that modulates LPS-mediated activation of JNK kinase activity, the method comprising:

providing a cell that harbors LPS and expresses a polypeptide comprising a CARD, NBS, or LRR domain of CARD-4;

exposing the cell to a test compound; and measuring JNK kinase activity in the cell;

wherein altered JNK kinase activity in the presence of the test compound compared to JNK kinase activity in the absence of the test compound indicates that the test compound is a candidate compound that modulates LPS-mediated activation of JNK kinase activity.

- 6. The method of claim 5, wherein the cell is infected with Shigella flexneri.
- 7. The method of claim 5, wherein the cell is infected with Salmonella typhimurium.
 - 8. The method of claim 5, wherein the cell is infected with Helicobacter pylori.

9. A method for identifying a candidate compound that modulates an LPS-induced immune response, the method comprising:

providing a cell that expresses a polypeptide comprising a CARD, NBS, or LRR domain of CARD-4;

introducing LPS into the cell;

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exposing the cell to a test compound; and

measuring oligomerization of the polypeptide in the cell;

wherein altered oligomerization of the polypeptide in the presence of the test compound compared to oligomerization of the polypeptide in the absence of the test compound indicates that the test compound is a candidate compound for modulating an LPS-induced immune response.

- 10. The method of claim 9, wherein the cell is infected with Shigella flexneri.
- 11. The method of claim 9, wherein the cell is infected with Salmonella typhimurium.
 - 12. The method of claim 9, wherein the cell is infected with Helicobacter pylori.
- 13. A method of modulating LPS-induced activation of NF-kB or JNK, the method comprising:

providing a cell that harbors intracellular LPS; and

contacting the cell with a compound that modulates expression or activity of CARD-4 in an amount sufficient to modulate LPS-induced activation of NF-kB or JNK in the cell.

14. A method of modulating an LPS-induced immune response in an individual, the method comprising:

selecting an individual comprising cells harboring intracellular LPS; and administering to the individual a compound that modulates expression or activity of CARD-4 in an amount sufficient to modulate an LPS-induced immune response in the individual.

- 15. The method of claim 14, wherein the individual is diagnosed as having a bacterial infection.
- 16. The method of claim 15, wherein the bacterial infection is a Shigella flexneri infection.

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- 17. The method of claim 15, wherein the bacterial infection is a Salmonella typhimurium infection.
- 18. The method of claim 15, wherein the bacterial infection is a *Helicobacter pylori* infection.
- 19. A method of treating or preventing a bacterial infection, the method comprising: selecting an individual having or at risk of having a bacterial infection; administering to the individual a compound that modulates expression or activity of CARD-4 in an amount sufficient to treat or prevent the bacterial infection.
- 20. A mouse whose genome comprises a disruption in an endogenous CARD-4 gene, wherein said disruption results in decreased expression or a lack of expression of said endogenous CARD-4 gene, thereby causing a decreased ability of the mouse to clear a Salmonella typhimurium or Helicobacter pylori infection.